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Outcome and Profile of Ventricular Septal Rupture With Cardiogenic Shock After Myocardial Infarction: A Report from the SHOCK Trial Registry

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OBJECTIVES We wished to assess the profile and outcomes of patients with ventricular septal rupture (VSR) in the setting of cardiogenic shock (CS) complicating acute myocardial infarction (MI).

BACKGROUND Cardiogenic shock is often seen with VSR complicating acute MI. Despite surgical therapy, mortality in such patients is high.

METHODS We analyzed 939 patients enrolled in the SHOCK Trial Registry of CS in acute infarction, comparing 55 patients whose shock was associated with VSR with 884 patients who had predominant left ventricular failure.

RESULTS Rupture occurred a median 16 h after infarction. Patients with VSR tended to be older ($p = 0.053$), were more often female ($p = 0.002$) and less often had previous infarction ($p < 0.001$), diabetes mellitus ($p = 0.015$) or smoking history ($p = 0.033$). They also underwent right-heart catheterization, intra-aortic balloon pumping and bypass surgery significantly more often. Although patients with rupture had less severe coronary disease, their in-hospital mortality was higher (87% vs. 61%, $p < 0.001$). Surgical repair was performed in 31 patients with rupture (21 had concomitant bypass surgery); 6 (19%) survived. Of the 24 patients managed medically, only 1 survived.

CONCLUSIONS There is a high in-hospital mortality rate when CS develops as a result of VSR. Ventricular septal rupture may occur early after infarction, and women and the elderly may be more susceptible. Although the prognosis is poor, surgery remains the best therapeutic option in this setting. (J Am Coll Cardiol 2000;36:1110–6) © 2000 by the American College of Cardiology

Ventricular septal rupture (VSR) is an uncommon but well-recognized mechanical complication of acute myocardial infarction (MI). Seen with anterior or inferior MI, it carries a poor prognosis. Advances in echocardiography, including Doppler and multiplane transesophageal imaging, now enable the reliable diagnosis, localization and quantification of intracardiac shunting in patients with VSR (1–3). Intra-aortic balloon pumping (IABP) provides temporary but effective early medical stabilization. The first successful surgical repair by Cooley et al. (4) established the role of surgical intervention, although surgery was restricted initially to long-term survivors (5–7). The recognized limitations of medical management (8,9), the availability of the IABP (10) and improvements in surgical technique and myocardial protection soon led to aggressive surgical management for most patients with VSR (11,12). Despite the increasing use of surgery and the reasonable long-term prognosis for most surgical survivors (13–15), most patients with VSR and cardiogenic shock (CS) do not survive (16–21). Precarious hemodynamic characteristics, the complexity of the surgery, ongoing ischemia and underlying left ventricular (LV) and right ventricular (RV) dysfunction are major contributors to this poor outcome.

The SHould we emergently revascularize Occluded Coronaries in cardiogenic shocK? (SHOCK) study was a prospective, multicenter registry and randomized trial of CS complicating acute MI. The randomized trial was restricted to patients with predominant LV pump failure; thus, those with a mechanical etiology of CS were excluded. Such patients were followed prospectively as part of the SHOCK Trial Registry, however. We wished to assess the effect of surgical repair on outcomes of VSR and the profile and outcomes of patients with CS complicating acute MI who did and did not suffer VSR.

METHODS

The design and inclusion criteria for the randomized SHOCK Trial have been previously published (22). Details

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Abbreviations and Acronyms

BP	= blood pressure
CAD	= coronary artery disease
CK	= creatine kinase
CS	= cardiogenic shock
ECG	= electrocardiogram, electrocardiographic
IABP	= intra-aortic balloon pump
LV	= left ventricular, left ventricle
MI	= myocardial infarction
MR	= mitral regurgitation
RV	= right ventricular, right ventricle
SHOCK	= SHould we emergently revascularize Occluded Coronaries in cardiogenic shocK?
TIMI	= Thrombolysis In Myocardial Infarction
VSR	= ventricular septal rupture

of the nonrandomized SHOCK Trial Registry are published in this supplementary issue of the *Journal* (23).

Study population. There were 1,190 patients overall in the SHOCK Trial Registry. Of these, CS was due to predominant LV failure in 884 patients and due to VSR in 55. These 939 patients form the basis of this report. In all, 573 (61%) were registered in 24 U.S. centers; 196 (21%) in five Canadian centers; 55 (6%) in four Belgian centers; and 115 (12%) in Australia, New Zealand or Brazil. The mode of VSR diagnosis (echo/LV angiogram/right heart catheterization) was not recorded in the individual case report forms.

Definitions. Predominant LV failure was identified as the etiology for CS shock in the absence of other major shock categories such as isolated RV failure; acute, severe mitral regurgitation (MR); VSR; tamponade/LV rupture; prior severe valvular heart disease; or iatrogenic shock. Recurrent ischemia was defined as rest angina or ischemic symptoms ≥5 min with ST-segment depression, T-wave inversion, or both, without cardiac enzyme elevation. The index MI was defined as the infarction that caused CS. Re-infarction was defined as 1) recurrent chest pain or ischemic symptoms ≥30 min and recurrent ST-segment elevation, new Q waves, or new left bundle branch block; 2) total creatine kinase (CK) at least twice the upper limit of normal and >25% or 200 U/mL over the previous value, with an elevated CK-MB level; or 3) a rise in CK-MB above the upper limit of normal after it had reverted to the normal range.

Statistical analysis. Continuous variables were summarized as mean ± SD or median (interquartile range), and categorical variables as percentages. We compared the characteristics and outcomes of the 55 patients with VSR with those of the 884 patients in the SHOCK Trial Registry whose CS was caused by predominant LV failure. The Fisher exact test was used to assess differences in dichotomous or unordered categorical covariates. The Wilcoxon rank-sum statistic was used to compare the distributions of continuous variables between the two groups. For normally distributed covariates, the Student *t*-test was used. Gender, age and weight differed between the two groups. To determine whether VSR was associated with mortality after adjusting for these covariates, a logistic regression model was fit. We also attempted to create univariable logistic regression models to determine risk factors for any surgical mortality associated with VSR, but the small sample size coupled with the low survival rate resulted in computational problems and unstable coefficients. Therefore, descriptive statistics similar to those outlined above were used to profile survival in this study group. A p value of <0.05 was considered statistically significant.

RESULTS

Patient characteristics. Table 1 displays the baseline characteristics for the 55 patients with VSR and the 884 patients with predominant LV failure. The VSR group tended to be older ($p = 0.053$) and had a higher proportion of women. They weighed less than the group with LV failure, and they were more likely to be transfer admissions. The VSR group had a better risk profile with less history of MI, smoking and diabetes mellitus. After adjusting for age and weight, female gender was an independent predictor of VSR (odds ratio, 2.32; 95% confidence interval, 1.08 to 4.96; $p = 0.031$).

Table 1. Baseline Characteristics of Patients With CS and VSR or Predominant LV Failure

	VSR (n = 55)	Predominant LV Failure (n = 884)	p Value
Age (yrs)	72 ± 10	69 ± 12	0.053
Female gender	58%	36%	0.002
Weight (kg)	71.5 ± 16.5 (35)	75.5 ± 16.1 (531)	0.046
Height (cm)	166.6 ± 10.4 (27)	168.4 ± 10.2 (451)	0.172
Transferred from elsewhere	64%	42%	0.002
History of hypertension	50%	52%	0.887
Diabetes	17%	33%	0.015
Smoking	35%	52%	0.033
History of elevated lipids	23% (26)	40% (443)	0.099
History of peripheral vascular disease	16% (32)	19% (560)	0.817
History of myocardial infarction	17%	40%	< 0.001
History of angioplasty	6%	7%	1.000
History of bypass surgery	2%	10%	0.052

Data presented are mean ± SD, or percentages. () = subgroup n when data was not available for the entire group.

Table 2. Hemodynamic Data for Patients With CS and VSR or Predominant LV Failure

	VSR (n = 55)	LV Failure (n = 884)	p Value
Right-heart catheterization	82%	64%	0.008
Heart rate (beats/min)	102 ± 23 (50)	95 ± 26 (832)	0.054
Systolic BP (mm Hg)	83 ± 13 (50)	88 ± 23 (833)	0.072
Diastolic BP (mm Hg)	52 ± 10 (45)	53 ± 17 (729)	0.304
Lowest systolic blood pressure (mm Hg)	71 ± 19 (36)	68 ± 17 (604)	0.065
Right atrial pressure (mm Hg)	18 ± 7 (25)	14 ± 7 (276)	0.009
Right ventricular systolic pressure (mm Hg)	56 ± 21 (10)	46 ± 20 (124)	0.091
Right ventricular diastolic pressure (mm Hg)	20 ± 6 (10)	15 ± 10 (118)	0.019
Pulmonary arterial systolic pressure (mm Hg)	48 ± 17 (27)	41 ± 13 (341)	0.050
Pulmonary arterial diastolic pressure (mm Hg)	23 ± 7 (26)	24 ± 8 (343)	0.295
Pulmonary capillary wedge pressure (mm Hg)	22 ± 9 (42)	24 ± 9 (534)	0.287
Left ventricular ejection fraction* (%)	40 ± 11 (16)	30 ± 13 (300)	0.002

Data presented as mean ± SD (number of patients with data available), or percentages. *Left ventricular ejection fraction same day as or after shock diagnosis. Hemodynamic measurements may have been obtained on support measures.
Beats/min = beats per minute.

Clinical profile of VSR patients. Most patients with VSR in the SHOCK Trial Registry (71%) had two systolic blood pressure (BP) measures <90 mm Hg recorded 30 min apart. In addition, 86% also required vasopressors to maintain systolic BP. Features of tissue hypoperfusion, as manifested by oliguria (<30 mL/h), were present in 81% of patients, and 83% had cold, diaphoretic extremities. The index MI was characterized by ST-segment elevation in 94% of the subjects, and >80% had ST-segment elevation when the diagnosis of shock was made. The median time from MI to diagnosis of VSR (n = 24) was 16 h (interquartile range, 1.8 h to 42.5 h). The median time from MI to VSR in 13 subjects given thrombolytic therapy was 18 h (5 h to 36 h). This did not differ significantly from the median time of 6 h (1 h to 89 h) among the 11 patients not given thrombolytics (p = 0.885).

Hemodynamic data (Table 2). The VSR patients more often underwent right-heart catheterization. Consistent with the presence of left-to-right shunting, patients with VSR had higher recorded right atrial, RV diastolic and pulmonary arterial systolic pressures. Left ventricular ejection fraction, measured on the same day or after shock, also was greater for the VSR group. The ratio of pulmonary to systemic blood flow was available for 10 patients with VSR; it averaged 2.6 ± 1.7.

Location of MI and angiographic findings. The electrocardiographic (ECG) location of MI was recorded in 52 of the 55 patients with VSR. The index MI was inferior in 26 patients, anterior in 22, anterior and inferior in three and apicolateral in one patient. Among those with inferior MI, five had lateral involvement, four posterior and one posterolateral. Similarly, four patients with anterior MI had involvement of the lateral wall, and another five had involvement of the apicolateral leads. In 11 of 32 patients with more detailed data (ECG, echocardiographic or hemodynamic), there was evidence of accompanying RV MI. Severe RV dysfunction was reported in four of these patients, whereas two patients each had moderate and mild impairment. Data were unavailable for three patients.

Coronary angiography was performed in 35 patients with VSR. The majority had single- or double-vessel coronary artery disease (CAD) (26% and 43%, respectively); a minority had left main (6%) or triple-vessel CAD (31%). The infarct-related artery was identified in 26 patients; it was most often the right (12/26, 46%) or left anterior descending coronary artery (11/26, 42%). The left circumflex artery was the culprit vessel in only three cases. All culprit vessels had >90% stenosis, and the overwhelming majority (22/26) exhibited Thrombolysis In Myocardial Infarction (TIMI) grade 0 or 1 flow. The median LV ejection fraction was 35% (interquartile range, 25% to 40%) in the 13 subjects who underwent ventriculography. Table 3 shows the angiographic findings of the VSR patients and those with

Table 3. Angiographic Findings in Patients With CS and VSR or Predominant LV Failure

	Current Study		Prior Study VSR*
	VSR	LV Failure	
Infarct-related artery††	(n = 26)	(n = 405)	
Right coronary	46%	28%	
Left anterior descending	42%	45%	
Left circumflex	12%	15%	
Severity of disease†			
	(n = 35)	(n = 518)	(n = 232)
0 vessel	0%	1%	0%
1 vessel	26%	21%	44%
2 vessel	43%	21%	36%
3 vessel	31%	57%	19%
Left main	6%	17%	NA
Ejection fraction (%)	35 (n = 13)	30 (n = 173)	

*Distribution of atherosclerotic coronary artery disase in previously reported series also shown for comparison (24). †Distribution differed significantly between the VSR and LV failure groups (p = 0.008). ††Culprit artery was similar for both groups (p = 0.184). Data presented are medians or percentages.
NA = not available.

Table 4. Management of Patients With CS and VSR or Predominant LV Failure

	VSR (n = 55)	LV Failure (n = 884)	p Value
Thrombolytic therapy	43.6%	34.5%	0.190
Intra-aortic balloon counterpulsation	74.5%	52.6%	0.002
Angiography	67.3%	61.3%	0.396
Angioplasty	9.1%	32.9%	< 0.001
Bypass surgery	40.0%	15.4%	< 0.001
Mechanical ventilation	74.5%	75.3%	0.873

Data presented are percentages.

predominant LV failure. Also included for comparison are pooled angiographic findings for 232 patients with VSR previously reported (24). The extent of CAD appeared to be greatest in the group with predominant LV failure, but the VSR patients in this report appeared to have more extensive CAD than those previously reported.

Treatment (Table 4). Intravenous thrombolytic therapy was given to 24 patients with VSR, beginning a median 2.9 h after MI onset (interquartile range, 1.9 h to 4.1 h). Vasopressor therapy was given to 97% (36/37) of VSR patients; 75% (41/55) were mechanically ventilated; and intra-aortic balloon pump (IABP) was inserted in 75% (41/55). Within 30 min of initiation of IABP support, the median systolic BP in the VSR group had increased from 81 mm Hg (interquartile range, 70 mm Hg to 92 mm Hg) to 102 mm Hg (87 mm Hg to 120 mm Hg) ($p < 0.001$). Angioplasty of the infarct-related artery was performed in five VSR patients, with an 80% procedural success rate (residual stenosis <50%). Three of the VSR patients undergoing angioplasty died without attempted VSR repair. Another went for surgical repair and later died, and one angioplasty patient survived to discharge. Thirty-one of the 55 VSR patients underwent surgical repair of the VSR (see following text). The time from MI to initiation of thrombolytic therapy was similar between VSR patients and those with predominant LV dysfunction. The VSR group was more likely to receive IABP support. Bypass surgery also was performed more often in the VSR patients, whereas angioplasty was used less often.

Outcomes. The overall in-hospital survival rate was only 13% (7/55) for patients with VSR, significantly lower than the 39% rate for patients with predominant LV failure ($p < 0.001$). Recurrent ischemia occurred more often in the VSR group (33% vs. 19%; $p = 0.05$). The rates of recurrent MI before shock onset did not differ (14% for the VSR group vs. 8%; $p = 0.226$). The median highest total CK was 1,188 U/L in the VSR patients and 1,923 U/L in the patients with LV failure patients ($p = 0.055$).

Surgery. Ventricular septal rupture repair was performed in 31 of the 55 patients, 21 of whom had concomitant coronary artery bypass grafting. Three patients had accompanying LV free-wall rupture; another had RV free-wall rupture; and three others underwent aneurysmectomy. The median perfusion time was 201 min (interquartile range, 158 min to 262 min; n = 16), and the median cross-clamp time was 130 min (interquartile range, 90 min to 164 min; n = 16). Of those undergoing bypass surgery, 90% had complete revascularization, though saphenous vein grafts were used exclusively. Overall mortality in the surgical group was 81% (25/31) and was uniform across centers enrolling in the SHOCK Trial Registry. Of the 24 patients not undergoing surgical VSR repair, only one survived. Patients undergoing surgical repair were younger (median 72 years vs. 77 years; $p = 0.04$) than their medically treated counterparts. We were unable to construct mortality models to predict the survival of VSR patients undergoing surgical repair, because of the small sample size (31 patients with only six survivors).

Table 5. Profile of Surgically Managed Patients With CS and VSR

	Survivors (n = 6)	Nonsurvivors (n = 25)
Age (yrs)	69 (6)	72 (25)
Female gender	50% (6)	60% (25)
Transferred from elsewhere	67% (6)	56% (25)
Prior myocardial infarction	17% (6)	8% (24)
Hypertension	33% (6)	58% (24)
Diabetes	0% (6)	21% (24)
Smoking	40% (5)	43% (21)
Anterior infarction	83% (6)	38% (24)
Inferior infarction	33% (6)	67% (24)
Apical infarction	50% (6)	13% (24)
Time from infarct onset to shock onset (h)	52 (5)	16 (20)
Time from infarct onset to VSR (h)	154 (4)	13 (11)
Time from shock onset to surgical VSR repair (h)	27 (5)	8 (17)
Heart rate (beats/min)	110 (6)	95 (21)
Systolic blood pressure (mm Hg)	94 (6)	85 (21)
Diastolic blood pressure (mm Hg)	55 (5)	51 (20)
Right atrial pressure (mm Hg)	13 (3)	17 (13)
Pulmonary arterial systolic pressure (mm Hg)	62 (3)	40 (14)
Pulmonary capillary wedge pressure (mm Hg)	23 (4)	21 (21)
Angiography	83% (6)	92% (25)
Bypass surgery	50% (6)	72% (25)

Data presented are medians or percentages (number of patients with data available). Beats/min = beats per minute.

Profile of survivors. Six of the overall seven survivors had VSR repair. Survivors of VSR repair (n = 6) are compared with surgical nonsurvivors in Table 5. Surgical survivors appear to have more anterior/apical MI and to have longer times from MI onset to shock onset, from MI onset to VSR onset, and from shock onset to VSR repair. Right atrial pressures appear to be lower in survivors, whereas pulmonary arterial systolic pressures were higher. To determine the factors associated with mortality in patients undergoing VSR repair, a number of logistic regression models were attempted. As mentioned, we could not construct a multi-

Table 6. Profile of Surgical Survivors With CS and VSR

	Survivor 1	Survivor 2	Survivor 3	Survivor 4	Survivor 5	Survivor 6
Age (yrs)	67	74	67	78	66	71
Gender	male	female	male	female	female	male
Transferred from elsewhere	no	yes	yes	no	yes	yes
Anterior infarction	yes	yes	yes	yes	yes	no
Inferior infarction	no	yes	no	no	no	yes
Time from MI onset to shock onset (h)	228	52	67	24	36	—
Time from MI onset to VSR (h)	236	—	89	219	36	—
Time from shock onset to VSR surgery (h)	11	27	139	200	—	5
Left-heart catheterization	yes	yes	yes	yes	no	yes
Angioplasty	no	no	no	no	no	no
Bypass surgery	yes	yes	no	no	no	yes
Systolic blood pressure (mm Hg)*	104	85	125	83	94	93
Diastolic blood pressure (mm Hg)*	—	50	55	42	58	66
Heart rate (beats/min)*	118	105	115	72	150	100
Pulmonary capillary wedge pressure (mm Hg)*	23	20	24	—	—	22
Pulmonary arterial systolic pressure (mm Hg)*	56	—	96	—	—	62
Pulmonary arterial diastolic pressure (mm Hg)*	25	—	—	—	—	22
Right atrial pressure (mm Hg)*	13	—	17	—	—	11

*While on support measures. Data presented are medians or percentages.
Beats/min = beats per minute; MI = myocardial infarction.

variate model for surgical mortality, because of the small number of survivors. As a result, a detailed profile of survivors is presented in Table 6. The sole in-hospital medical survivor was a 35-year-old male smoker with an anterior MI. The time from MI onset to shock onset was 7.35 h, and angioplasty was performed. The highest CK was 12,010 U/L; in-hospital ejection fraction was 43%; and he was diagnosed with VSR 48 h after MI onset.

Comparison with acute severe MR. Ninety-eight patients with CS secondary to acute severe MR were part of the SHOCK Trial Registry (25). Selected variables from this group are compared with our study group in Table 7. Patients with VSR have less prior MI and were less likely to be diabetic. Mean right atrial pressure was higher in the VSR setting, but pulmonary capillary wedge pressure and LV ejection fraction were similar. Patients with VSR had poorer in-hospital survival compared with patients having severe MR.

DISCUSSION

This SHOCK Trial Registry report describes a large series of patients with acute MI complicated by VSR and CS. By design, patients with VSR in the absence of shock are not included in this registry. All patients were managed in tertiary cardiac-care units. We observed a 87% mortality, and this very poor outcome was seen across all participating centers.

Demographic characteristics. Many of the observations in our study confirm previous reports. Ventricular septal rupture most often complicates a first MI, which may be anterior, apical, or inferior in location. The infarct-related vessel is usually severely stenosed with TIMI grade 0/1 flow.

Table 7. Patient Characteristics: VSR vs. Acute Severe MR in SHOCK Trial Registry Patients

Characteristic	VSR*	Severe MR*	p Value
n	54	97	
Mean age (yrs)	71.7 ± 10.0	69.8 ± 10.1	0.253
Female	57.4%	51.6%	0.502
History of hypertension	51.0%	59.0%	0.385
History of MI	15.1%	33.0%	0.020
Diabetes	17.0%	33.7%	0.035
Smoking	35.6%	46.0%	0.271
Anterior MI	49.0%	34.5%	0.106
Median MI to CS (hours)	15.8	13.2	0.601
Heart rate (beats/min)	101.6 ± 22.3 (49)	97.1 ± 24.1 (94)	0.246
Pulmonary capillary wedge pressure (mm Hg)	22.3 ± 8.5 (42)	22.1 ± 7.5 (76)	0.839
Right atrial pressure (mm Hg)	18.0 ± 7.2 (25)	14.1 ± 12.5 (42)	0.002
LV ejection fraction (%)†	40.6 ± 11.0 (17)	38.7 ± 17.2 (58)	0.281
In-hospital survival	13.0%	45.4%	< 0.001

*One patent who had both MR and VSR was excluded from this analysis; †Left ventricular ejection fraction obtained at anytime during the shock hospitalization.
() = Subgroup n.

The absence of collateral flow to the infarcted area creates a milieu for extensive transmural myocardial necrosis, predisposing to rupture.

The majority of our patients with rupture were female. This differs from the male predominance reported in many surgical (14,15,17,19,20,26) and post-mortem series (27,28). The mean age in our study (72 years) is greater than that previously reported. It appears consistent with the aging of the population in general and the increased risk of rupture with advancing age. Age also may contribute to the extent of angiographic CAD, which appears to be more severe than previously reported. The presence of CS in all our patients may also account for the severity of CAD, but the VSR group had less extensive CAD than those whose shock was due to predominant LV failure.

Timing of VSR. The median time from MI onset to VSR in our 24 subjects for whom timing data were recorded is shorter than the three to eight days reported in the literature (28). The universal presence of CS may have contributed to this discrepancy. Early reperfusion strategies appear to have decreased the incidence of cardiac rupture, free-wall rupture, and VSR (29,30). There is concern, however, that thrombolytic therapy may accelerate rupture in certain patients (31). This is not supported by our observation that there was no difference in time to VSR between patients who did and did not receive thrombolytic therapy.

A bias toward later VSR in some surgical series also may explain the discrepancy in VSR timing, and early death from VSR before surgical repair may have led to under-reporting in some series. Statistical representation may play a role because the timing of post-infarction VSR does not appear to be normally distributed; rather, it occurs either early or relatively late after MI. Thus the *mean* time to VSR may misrepresent the timing of rupture. In this situation, a few cases of VSR that occur remotely from the MI may markedly skew the overall reported time of VSR.

Prior surgical experience. Surgical intervention plays a pivotal role in the management of unstable patients with VSR after MI. Although a delay in intervention appears to be associated with improved surgical results, a high early mortality makes this approach untenable. Previous case series, with exceptions, have been single-center, retrospective, longitudinal analyses. Many have reported surgical survival alone, and patients with CS are often underrepresented. Gaudiani et al. (14) reported a 90% mortality for patients requiring operation within 24 h of VSR. In the series of Loisance et al. (16), 19 of 57 patients died before surgery was attempted, and the 14 patients who made it to surgery with CS had a 71% operative mortality. Similarly, Radford et al. (17) reported a 73% (8/11) surgical mortality at Massachusetts General Hospital, and reports from the Mayo Clinic (20) have been similarly disappointing. By contrast, Komeda et al. (13) reported a mortality of only 20% in 15 patients with VSR and CS, and Skillington et al. (32) reported a 39% mortality in 41 such patients. A more recent update reported a 52% mortality in 22 subjects with VSR, MI and CS (33).

Surgical outcomes in the present study. Advances in surgical technique and myocardial preservation have improved outcomes for most patients with VSR complicating acute MI (34). In addition, predictors of survival have been identified (35,36). Yet, as illustrated by our data, the subgroup with CS does poorly: only 7 of our 55 subjects with VSR and CS survived. Because of the small number of survivors, we were unable to develop a model for survival, but our observations confirm some previous findings. Even in this population with CS, patients with anterior MI and apical VSR do better than those with inferior MI (21), probably because the latter are usually anatomically complex, surgically more challenging and have greater RV infarction and dysfunction. In addition, we saw no additional benefit of concomitant bypass surgery in this small sample: of our six survivors, two had VSR repair alone.

The times from MI onset to shock onset, from MI onset to VSR, and from MI onset to surgical VSR appear to be longer for survivors than for nonsurvivors. These observations are based on a very small number of survivors but are consistent with previous reports in the literature. The association of survival and later surgery may be explained by survivorship bias. These data are not adequate to produce clear conclusions about the timing of surgery for VSR, considering the small number of survivors and of selection bias. However, our disappointing results should not be viewed as an excuse to postpone treatment of patients with VSR. Our population included only patients who had developed shock. Patients who had early VSR repair before the onset of shock were excluded from our study. Our poor outcomes once shock has developed would tend to support the Class I indication for urgent early surgery before shock onset when VSR complicates MI (37). Delaying surgery to permit infarct healing risks the development of shock, which is unpredictable and associated with poor surgical prognosis. Despite the poor outcome, surgery in this setting remains the best therapeutic option. Surgery may be near futile for elderly patients with inferior MI complicated by shock, VSR and other comorbidity.

Conclusions. This report from an international, prospective registry of patients with clinical CS highlights the dismal outcome of those with VSR complicating acute MI. Patients with VSR were more often elderly and female and less often had multi-vessel CAD, compared with patients whose shock was due to predominant LV failure. In most patients, VSR occurred early after a first, index MI. Medical management resulted in almost 100% mortality, and surgical outcome was poor once CS developed.

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